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INSIDE

Potential Role for Leukotriene Inhibitors in Asthma Management

ABSTRACT & COMMENTARY

Source: Silverman RA, et al. Zafirlukast treatment for acute asthma: Evaluation in a randomized, double-blind, multicenter trial. *Chest* 2004;126:1480-1489.

THE OBJECTIVE OF THIS STUDY WAS TO DETERMINE THE EFFECT of zafirlukast, an oral leukotriene receptor antagonist, in ED patients presenting with acute exacerbations of asthma. Adult patients presenting with asthma exacerbations were enrolled provided that they had an FEV1 less than 70% of predicted after an initial dose of nebulized albuterol 2.5 mg. Patients who had serious comorbidities or had received oral corticosteroids or leukotriene-modifying drugs within two weeks of presentation were excluded. All study subjects received repeated doses of nebulized albuterol and prednisone 60 mg orally. Patients were randomized, in a double-blinded fashion, to experimental and control groups. Those in the experimental group received either zafirlukast 160 mg (Z160) or zafirlukast 20 mg (Z20) orally; those in the control group received a matching placebo.

All patients remained in the ED for 4 hours, and then a disposition decision was made. Patients not ready for discharge, per the treating physicians' assessment, were discontinued from the study protocol. Those patients in the zafirlukast group who were suitable for discharge at 4 hours received zafirlukast 20 mg BID for 28 days, while those in the control group received a matching placebo. Discharged patients also were provided with prednisone 20 mg BID, an albuterol inhaler to use as needed, and instructions to resume their usual asthma regimen. The primary study endpoint was time to relapse, defined as the need to return to the ED or make an unscheduled office visit due to exacerbation of asthma before the end of the 28-day study period. A secondary endpoint was the need for extended ED care after the initial four-hour treatment period.

The authors enrolled 641 patients with approximately equal numbers in the control and zafirlukast groups. Within the zafirlukast group, approximately equal numbers were assigned to the Z20 and

Z160 arms. Baseline demographics, spirometry values, and dyspnea scores were similar between the groups. Treatment beyond 4 hours was required in 17% of patients in the group receiving Z20, 10% of those receiving Z160, and 15% of control patients (odds ratio of 0.54 for Z160 vs. placebo, difference not significant for Z20 vs. placebo). Compared with the placebo group, dyspnea scores at 4 hours were significantly better in the Z160 group, but not in the Z20 group. Relapse within 28 days of ED evaluation was noted in 24% of patients discharged with zafirlukast, vs. 29% of control patients (5% risk reduction, $p < 0.05$). The relapse rate did not differ according to the dose of zafirlukast initially administered in the ED.

Adverse events were uncommon, mild, and did not differ significantly between the treatment groups. The authors concluded that treatment with zafirlukast 160 mg in the ED and zafirlukast 20 mg BID after discharge reduces the need for prolonged ED care and the rate of relapse after ED discharge. Four of the study authors are employees of the manufacturer of zafirlukast. ❖

COMMENTARY BY DAVID J. KARRAS, MD, FAAEM, FACEP

This study represents the growing interest in adding disease-controlling agents to the medical regimen of asthma patients treated for acute disease exacerbations. Agents such as inhaled corticosteroids and oral leukotriene receptor antagonists clearly help many patients avoid acute asthma exacerbations; but it is less clear whether they have a role in preventing short-term asthma relapses following an exacerbation. Studies of inhaled corticosteroids started upon ED discharge have yielded conflicting results.[1,2] It is likely that the potency of oral corticosteroids, now administered routinely after ED discharge, obviates the local disease-modifying effects of inhaled medication.

Studies of the leukotriene receptor antagonists in acute asthma exacerbations have had similarly mixed results. One study found a benefit to intravenous montelukast in patients with acute disease.[3] Given that placement of an IV is no longer necessary for the majority of patients treated for asthma exacerbation, an effective oral supplement to standard asthma therapy would have obvious advantages.

The benefit to zafirlukast noted in this study is somewhat limited. Treatment with high-dose zafirlukast reduced by 34% the relative rate of treatment beyond 4 hours, but the absolute risk reduction was only 5%. We are not provided with information regarding how many patients actually required hospitalization, only the number requiring therapy beyond 4 hours.

The benefit of zafirlukast after ED discharge was similar, reducing the relative relapse rate by 18% but the absolute rate by only 5%. These reductions in relative risk are not small, but they do not translate into dramatic ifferences in outcome; relatively few patients have poor outcomes.

It is important to remember that the study was funded by the manufacturer of the study product, and four representatives of the company—including, apparently, the biostatistician—are listed as authors. This does not negate the importance of the study, and indeed the study design does not appear biased toward the product. However, the conclusions would be far more convincing if the authors did not have an overwhelming interest in presenting the data in the most favorable light.

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Emergency Medicine Alert, ISSN 1075-6914, is published monthly by Thomson American Health Consultants, 3525 Piedmont Rd., NE, Bldg. 6, Suite 400, Atlanta, GA 30305.

Vice President and Group Publisher: Brenda Mooney.
Editorial Group Head: Glen Harris.
Managing Editor: Martha Jo Dendinger.

GST Registration Number: R128870672.

Periodicals postage paid at Atlanta GA 30304. **POSTMASTER:** Send address changes to ***Emergency Medicine Alert***, P.O. Box 740059, Atlanta, GA 30374.

Back issues: $48. One to nine additional copies, $234 each; 10 to 20 additional copies, $173 each.

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United States: $299 per year (Resident rate: $144.50)
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This publication does not receive commercial support.

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Self-administered Antidysrhythmics for Perceived Episodes of Atrial Fibrillation

ABSTRACT & COMMENTARY

Source: Alboni P, et al. Outpatient treatment of recent-onset atrial fibrillation with the "pill-in-the-pocket" approach. *N Engl J Med* 2004;351:2384-2391.

Depending on the clinical presentation, there is currently a wide range of treatment options for the ED management of patients with atrial fibrillation (AF). This Italian study evaluated the feasibility, efficacy, and safety of an outpatient pill-in-the-pocket approach, whereby patients who were treated successfully in the ED with flecainide or propafenone were provided the same medication for self-administration and outpatient use for recurrent AF episodes.

Investigators studied 268 patients presenting to the ED who were diagnosed with AF of recent duration (fewer than 48 hours of onset) and treated with either oral flecainide (300 mg or 200 mg if fewer than 70 kg) or propafenone (600 mg or 450 mg if fewer than 70 kg). Of those patients, 210 patients had restoration of sinus rhythm either in the ED or as an inpatient and were enrolled in the study. The patients were discharged with the same medication (74 with flecainide; 136 with propafenone) that was used in the study and told to self-administer the drug as an outpatient in the event of recurrent heart palpitations.

During the follow-up period (mean duration of 15 months), 165 patients had a total of 618 episodes of palpitations. In 569 of those episodes, patients administered the medication as directed (mean time to self-admini tration was 36 minutes); patients noted successful resolution of their symptoms within 6 hours (mean time 113 minutes) in 534 episodes (94%). In the remaining 35 episodes, treatment was unsuccessful or resolution occurred after 6 hours; and 26 of those resulted in a return visit to the ED. An additional five ED visits occurred in the 49 episodes where self-treatment was not attempted by the patient. Adverse effects occurred in 12 patients (7%) and included one episode of rapid ventricular response and 11 cases of noncardiac side effects.

The investigators retrospectively compared palpitation episodes, ED visits, and hospitalizations before and after the study in the 210 patients enrolled. For the group, mean episodes per month were the same (59.8 vs. 54.5 before and after the study respectively), but ED visits per month markedly decreased (45.6 vs. 4.9 respectively) as did hospitalizations per month (15.0 vs. 1.6 respectively). Twenty-seven patients did drop out of the study because either the drug was ineffective or the patient was switched to prophylactic treatment due to frequent recurrences.

Based on their findings, the authors conclude that the pill-in-the-pocket approach of self-administration of either flecainide or propafenone for episodes of atrial fibrillation is feasible, safe, and effective for a select group of patients who present to the ED with dysrhythmia. In addition, this approach appears to reduce repeat ED visits and hospitalizations. ❖

COMMENTARY BY TED CHAN, MD, FACEP

The management of patients who present to the ED with new-onset atrial fibrillation continues to evolve. Flecainide and propafenone are rapidly-acting class IC antidysrhythmics that have efficacy in converting AF of recent onset to sinus rhythm.[1] The study investigators found that patients who are treated in the ED with these medications can treat themselves safely and effectively for recurrent AF episodes on an outpatient basis.

Many points are worth emphasizing with this study. First, both flecainide and propafenone were used in this study and no data are provided actually comparing the two drugs to determine if one was better. Second, patients self-administered the medication with the onset of palpitations, but there are no data confirming the presence of AF. Thus, patients took the medications based on nonspecific symptoms that may or may not have been the dysrhythmia.

Third, and most importantly, exclusion criteria for this study were numerous and included the presence of common cardiac conditions (e.g., ischemic heart disease, cardiomyopathy, heart failure history, left ventricular dysfunction, preexcitation, bundle-branch block, second and third degree atrioventricular block) and noncardiac conditions (e.g., liver or renal dysfunction). As a result, at one site, only 12% of patients presenting to the ED with AF actually were eligible for the study. Accordingly, application of this

pill-in-the-pocket approach must be limited to a very select AF patient population presenting to the ED.

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Utility of Serum Electrolyte Determination in Dehydrated Pediatric Patients

ABSTRACT & COMMENTARY

Source: Wathen JE, et al. Usefulness of the serum electrolyte panel in the management of pediatric dehydration treated with intravenously administered fluids. *Pediatrics* 2004; 114:1227-1234.

A SERUM ELECTROLYTE PANEL (SEP) OFTEN IS obtained in the management of pediatric patients dehydrated due to gastroenteritis and needing intravenous fluids. The usefulness of this practice was evaluated prospectively in a convenience sample of 182 children, 2 months to 9 years of age, seen in a pediatric emergency department (PED) with an observation unit. Outcome measures included frequency of an abnormal SEP, changes in management as a result of the SEP, relationship of SEP results to patient disposition, and unscheduled return visits (URV). The ability of physicians to predict an abnormal SEP also was studied.

One hundred-eleven patients (61%) had mild dehydration, 55 (30%) were moderately dehydrated, and 16 (9%) had severe dehydration. One hundred sixty-five patients (91%) were discharged from the PED, with seven (4%) having URVs. Seventeen patients (9%) were admitted with two having URVs. Eighty-eight patients (48%) had one or more abnormal SEP values, most commonly from low bicarbonate levels, high blood urea nitrogen (BUN) levels, hypoglycemia, hypokalemia, and hypernatremia.

Significantly low serum bicarbonate levels (less than 13 mmol/L) were noted in those children younger than one year, as well as those with a higher estimated degree of dehydration, and more diarrhea. Low serum bicarbonate and glucose levels were associated most commonly with changes in clinical management. In only 19 (10%) patients did an abnormal SEP change management. Physicians were able to predict only 58% of clinically significant SEP results. ❖

COMMENTARY BY RAEMMA LUCK, MD

The American Academy of Pediatrics (AAP) practice parameter on the management of acute gastroenteritis recommends that an SEP be considered in patients with moderate to severe dehydration and those needing intravenous fluids.[1] The authors should be commended for raising some questions regarding the value of routinely obtaining an SEP in dehydrated patients needing intravenous fluids. The results of the study showed that 48% of patients had one or more abnormal SEP results, but in only 10% of cases did this change the management. The authors did not break down the clinically significant study results as to the degree of dehydration. Breaking down the results as to which groups have clinically relevant values would provide more useful information to the physician and perhaps limit laboratory testing to a certain subset of patients.

The authors have pointed out that infants younger than one year had lower serum bicarbonate levels despite similar degrees of dehydration. They also were more likely to receive additional intravenous fluids in the observation unit. Correlating significant SEP results with the age group (e.g., younger than or older than one year), in addition to the degree of dehydration, would refine what we know and add depth to the study.

The majority of patients in the study had only mild dehydration (61%), yet they were receiving intravenous fluids. The AAP still recommends oral rehydration therapy for mild to moderate dehydration.[1] However, there has been a documented gap between this clinical guideline and what is done currently in clinical practice.[2] A survey revealed that physicians are more likely to use intravenous therapy in cases where vomiting is the major symptom.[2] The use of adjunctive medications, such as ondansetron, has been shown to decrease vomiting and also decrease the need for admission.[3,4] Oral ondansetron, in conjunction with oral rehydration, might obviate the need for intravenous fluids in some patients with mild dehydration but continued vomiting.[3] It also has been shown that parenteral ondansetron was effective in reducing vomiting as well as reducing hospitalization in those patients with gastroenteritis who need intravenous fluids.[4] This would be useful especially in busy EDs where beds are tight and in those EDs without observation units. Ability to tolerate fluids also would decrease the number of URVs, which in this study was nearly 5% of the total cases.

Clinicians were able to predict only 58% of significant SEP values. One of ten patients had results that altered management. Hence, once a decision is made to give intravenous fluids, the practice of obtaining an SEP, consistent with AAP recommendations, is here to stay.

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Special Feature

Acute Compartment Syndrome

By Andrew D. Perron, MD, FACEP, FACSM

Introduction

Compartment syndrome is a serious limb- and life-threatening complication of extremity trauma. Fractures, crush injuries, burns, and arterial injuries all can result in an acute compartment syndrome. It develops when there is increased pressure within a closed tissue space, such as muscle compartments bound by dense fascial sheaths.[1] This increased pressure compromises blood flow through vessels supplying the contained muscles and nerves. Compartments in the arm and leg are most vulnerable to this syndrome, but virtually any muscle group surrounded by fascia is at risk. Frequently cited locations for compartment syndrome other than the lower leg include the hand, forearm, shoulder, back, buttocks, thigh, and foot.[2-4]

The incidence of compartment syndrome in the emergency department (ED) is unknown. Approximately three-quarters of cases are associated with fractures,[5] and tibia fracture has the highest association.[6-7] Early diagnosis and treatment is of the utmost importance; delay can lead to tissue necrosis, and ultimately severe, permanent disability, as with Volkmann's ischemic contracture.

Pathophysiology

Compartment syndrome results when there is increased pressure within a closed tissue space that compromises blood flow to muscles and nerves. The increase in pressure can result from external compression of the compartment (e.g., circumferential cast or burn eschar) and/or volume increase within the compartment (e.g., hematoma or edema). The pathophysiology of compartment syndrome also can involve local hydrostatic and osmotic pressure conditions within the compartment. When the intracompartmental pressure increases above a specific level due to the above factors, perfusion is impaired, resulting in disruption of metabolic processes. Cell wall membrane integrity is compromised, leading to cytolysis and release of osmotically active cellular contents, drawing additional fluid into the interstitial space.[8] The net effect is increased pressure and further impairment of perfusion to the compartment and distal structures in that vascular distribution. This ultimately can lead to a compromise of the circulation and/or nerve conduction as well as irreversible muscle injury, contractions, loss of limb, myoglobinuria, renal failure, and even death.[9-10]

Common fractures associated with compartment syndrome are tibial fractures, supracondylar and humeral shaft fractures, and forearm fractures. Crush injuries to the hand or foot, with or without associated fractures, are also at risk.

Diagnosis

Clinical Presentation. A high suspicion remains the cornerstone of diagnosis. A traditional hallmark element in the history is pain disproportionate to the mechanism of injury in an awake, neurologically intact patient. Unfortunately, many patients at risk are injured severely or impaired, and cannot relate such symptoms.

The clinical signs of compartment syndrome often are remembered by using the mnemonic of the five Ps: pain, paresthesia, paresis, pallor, and pulses. Pain, especially disproportionate pain, often is the earliest sign, but the loss of normal neurological sensation is the most reliable sign.[9,11] On physical examination, palpation of the compartment in question may or may not demonstrate swelling or a tense compartment. In the awake, intact patient, active or passive range of motion in the affected limb will elicit significant pain. Decrease or loss of two-point discrimination also can be an early finding of compartment syndrome.[9,11] Clinical findings also can include shiny, erythematous skin overlying the involved compartment (described as a "woody" feeling),

and excessive swelling. A thready or diminished pulse is not a reliable sign. Intracompartmental tissue pressure is usually lower than arterial blood pressure, making peripheral pulses and capillary refill poor indicators of blood flow. Patients with a low diastolic blood pressure are more susceptible to compartment syndrome.[12] There are no distinguishing radiographic findings associated with compartment syndrome, but the fractures mentioned above (e.g., supracondylar and humeral shaft fractures, tibial fractures, and forearm fractures) should trigger consideration of this syndrome.

Determination of Compartment Pressures. The diagnosis of compartment syndrome is based primarily on determination of the intracompartmental pressure. There is some debate in the literature regarding what pressure level mandates fasciotomy. Some authors base recommendations on absolute compartment pressure,[9,13,14] while others feel the pressure is meaningful only as it relates to the mean or diastolic blood pressure.[15] Most literature, however, is based on absolute pressure within the compartment. Normal tissue pressure ranges between 0 and 10 mmHg. Capillary blood flow may be compromised at pressures greater than 20 mmHg. Muscle and nerve tissue is at risk for ischemic necrosis at pressures greater than 30 to 40 mmHg.

Several techniques are available for intracompartmental pressure determinations, each with advantages and disadvantages. They include the Stryker or Ace pressure monitors, the needle technique, the wick catheter, and the slit catheter. The Stryker and Ace monitors are used most frequently, and largely have replaced the other methods due to their ease of use and reproducible results. These monitors are self-contained, battery-powered pressure transducers. The other techniques have the advantage that they can be performed with items that are readily available in every ED. Descriptions of these techniques are beyond the scope of this article.

Regardless of the method used, the skin should be prepped with an antiseptic solution and infiltrated with local anesthesia at the prospective site. The site of pressure measurement is important. Heckman, et al[16] reported that failure to measure tissue pressure within a few centimeters of the zone of peak pressure might result in serious underestimation of the maximum compartment pressure. Their results suggest that measurements should be taken at the level of the fracture as well as at locations proximal and distal to the fracture to determine reliably the locations of highest tissue pressure, and that the highest pressure be used in the decision-making process.

Pulse oximetry has been advocated falsely as a simple noninvasive indicator of vascular compromise. Mars et al found that at clinically significant pressures, the test had a sensitivity of approximately 40%. Hence, with a greater than 50% risk of a false-negative result, pulse oximetry is *not* recommended in the detection of elevated compartmental pressure.[17]

Treatment

The goal of treatment is to decrease tissue pressure, restore blood flow, and minimize tissue damage and related functional loss. External pressure from casts or dressings should be alleviated immediately. It has been shown that if a cast is bivalved, the compartment pressures may decrease as much as 55%, and if completely removed, the pressure may decrease as much as 85%.[6,12] The limb should be elevated to the level of the heart to promote arterial blood flow but not decrease venous return.

Acute compartment syndrome is a surgical emergency. Fasciotomy is the definitive therapy and should be performed as soon as possible. Delays of more than 24 hours can have devastating consequences, as outlined above. According to Mabee,[1] absolute indications for fasciotomy are: 1) clinical signs of acute compartment syndrome, 2) raised tissue pressure greater than 30 mmHg in a patient with the clinical picture of compartment syndrome, and 3) interrupted arterial circulation to an extremity for greater than four hours. If performed within 12 hours of symptom onset, fasciotomy can prevent most ischemic myoneural deficits.[18] Most studies report that between 1% and 10% of patients with acute compartment syndrome will develop Volkmann's ischemic contracture.[2] ❖

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Physician CME Questions

11. In the study by Alboni et al, exclusion criteria for outpatient pill-in-the-pocket treatment of recent-onset atrial fibrillation included all of the following, *except:*
a. preexcitation.
b. ischemic heart disease.
c. heart failure.
d. atrial fibrillation of less than 48 hours duration.
e. renal dysfunction.

12. The _____ fracture classically is associated with compartment syndrome.
a. supracondylar
b. hamate
c. fifth metatarsal
d. patella

13. Normal tissue compartment pressures range from _____ mmHg.
a. 30-40
b. 20-30
c. 10-20
d. 0-10

14. Low pulse oximetry readings distal to the affected compartment are sensitive indicators of compartment syndrome.
a. True
b. False

15. In patients with acute asthma exacerbations, treatment with zafirlukast may result in:
a. diminished need for oral corticosteroid therapy on discharge.
b. a lower relapse rate within 28 days of discharge.
c. a lower rate of hospitalization.
d. greater dyspnea within 4 hours of treatment.

16. In the study by Walthen and colleagues, serum electrolyte results altered management in approximately ____ of pediatric patients with dehydration.
a. 5%
b. 10%
c. 15%
d. 20%

Answers:
11.d
12.a
13.d
14.b
15.b
16.b

CME Objectives

To help physicians:
- Summarize the most recent significant emergency medicine-related studies;
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Typical LBBB? LVH? Acute MI?

By Ken Grauer, MD

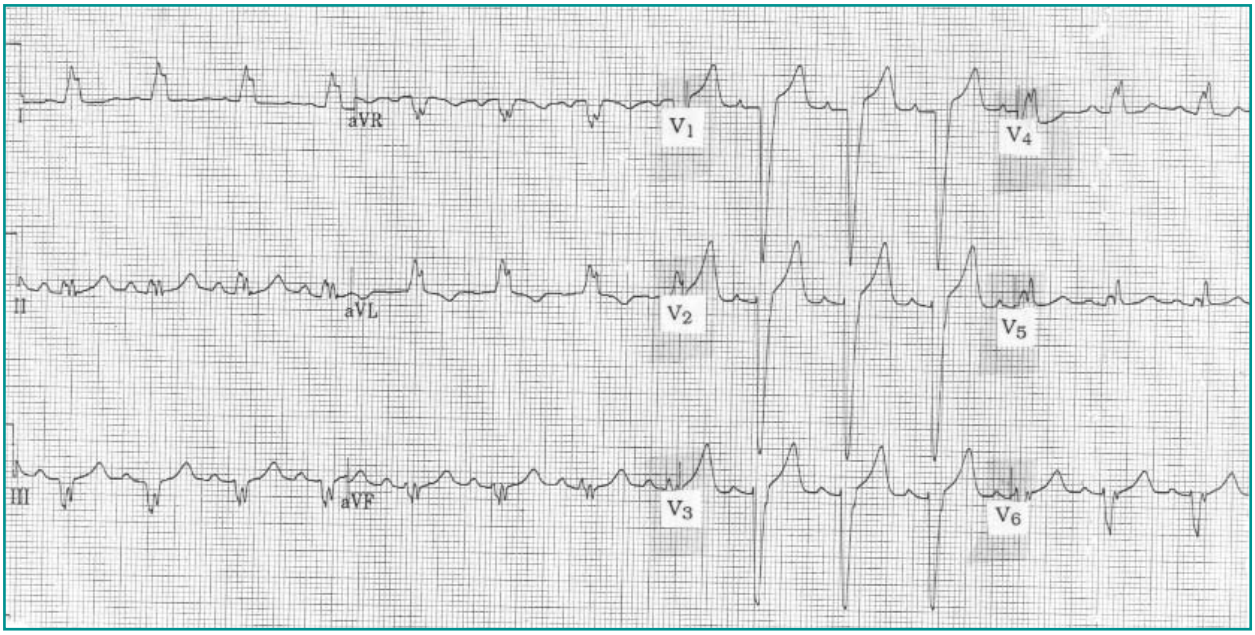

Figure: 12-lead ECG obtained from a 63-year-old woman with a history of hypertension, heart failure, and atypical chest pain.

Clinical Scenario: The electrocardiogram (ECG) in the figure was obtained from a 63-year-old woman with a history of hypertension, heart failure, and atypical chest pain. The ECG shows normal sinus rhythm at a rate of 85 beats/minute. The QRS complex is widened. Does it indicate a typical left bundle-branch block (LBBB)? Would you interpret this tracing as suggestive of left ventricular hypertrophy (LVH) or acute myocardial infarction (MI)?

Interpretation: The first point to make about this 12-lead ECG relates to the bizarre progression of QRS morphology in the precordial leads. It makes no anatomic (or physiologic) sense for the QRS complex to alternate from almost total negativity (in leads V_1, V_2, V_3)—to total positivity (in leads V_4, V_5)—and then abruptly back to near total negativity in lead V_6. Instead, we strongly suspect misplacement of several precordial leads. Most likely the QRS complex seen in lead V_6 really should appear in lead V_4 — and the complex in lead V_4 should appear in lead V_6. Were this the case, then this patient would manifest the typical pattern of complete LBBB (predominantly negative QRS in lead V_1; monophasic R wave with or without a notch in leads I and V_6). A repeat ECG is, of course, needed to verify our suspicion.

The diagnosis of LVH cannot be made by the usual criteria in the presence of complete LBBB because the conduction defect dramatically alters the usual sequence (and therefore QRS morphology) of ventricular activation. However, several relevant points relating to LBBB still can be made. First, most patients with complete LBBB have underlying heart disease. Simply the presence of LBBB identifies a high prevalence group of individuals who statistically are likely to have heart disease predisposing to ventricular hypertrophy (note the history of the 63-year-old woman in this case). In the presence of underlying heart disease and complete LBBB, the ECG finding of very deep S waves (of more than 25-30 mm) in leads V_1, V_2, and V_3 makes it highly likely that the patient also has LVH. However, nothing can be said about the presence or absence of myocardial infarction (old or acute) from interpretation of the typical LBBB pattern seen here. ❖

In Future Issues: Supracondylar fractures